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Editorial

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Identification of Protease Inhibitor-Caspase-6

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Abstract

Caspase-6 is a cysteine protease that plays important roles in axonal degeneration, programmed cell death, and development. The excess neuronal activity of Caspase-6 is associated with age-dependent cognitive impairment and Alzheimer disease neuropathology. The activation of the Caspase-6, aspartate-specific cysteinyl protease is proposed as an Huntington's disease and early pathogenic event of Alzheimer disease (AD). Caspase-6 inhibitors could be useful against these neurodegenerative diseases but most of the Caspase-6 inhibitors is used for the study of in vitro, show acute liver toxicity in humans.

Keywords

Caspase-6; Gene; DNA; Cysteine protease; Protease Inhibitor

History of Caspase-6

Caspase-6 was identified in 1995 from human Jurkat T lymphocytes using a PCR based approach. Initially named Mch2or 'mammalian Ced-3 homologue' the gene which known as caspase-6 since the introduction of the systematic nomenclature, Early studies identified caspase-6 as a pro-apoptotic executioner caspase that is activated by Caspase-3 and that cleaves Lamin A the first described caspase6

Roles of Caspases in Apoptosis and Inflammation Structural proteins: The common morphological features of apoptotic cells can be attributed to changes in structural proteins. Lamins, which maintain structural integrity of the nuclear envelop that are cleaved in many apoptotic cells.

Probably Caspase-6 is the primary lamin-cleaving caspase, since its substate specificity matches the site cleaved during apoptosis, Both inhibitor studies in apoptosing cells and *in vitro* cleavage assay suggest that fodrin is cleaved by caspases . Caspases is also having a role in regulating apoptotic plasma membrane changes resulting in phagocytic removal of apoptotic cells.

Caspase-6 in Health and Disease:

Caspases, initially identified as a family of Protease regulating cell death, have been found to have nonapoptotic functions as well. Some family members are critical for mediating Programmed cell death in development. After development, caspases are down regulated in the nervous system, but they continue to perform important nonapoptotic functions that are relevant for synaptic plasticity and neurogenisis. In neurodegenerative diseases, where neuronal death is an outstanding feature, there is an increase in caspase activity. The specific caspase death pathways leading to death have still not been fully clarified, dysfunction and despite .At present the current knowledge of caspase activation and activity pathways, functions of Caspases in the nervous system ,the current tools for examining caspases in health and in disease.

Alzheimer's Disease, the most common neurodegenerative disorder, and cerebral ischemia, the most common cause of neurologic death, are used to illustrate our current understanding of death signaling in neurodegenerative diseases.For better understanding of caspases function in health and disease they provide appropriate specific targets for the development of therapeutic interventions for these diseasesis a statistically valid method which make the best use of all available information.

Caspase- 6 in Mitochondria

Caspases are a family of Cystein process that cleave proteins following aspartic acid residues. These proteases exist in a hierarchy with upstream Caspases 2,8,9,10 and downstream caspases 3,6,7. Caspases are synthesized as largely inactive procaspases. Upstream procaspases, which are monomeric and they become activated when forced to dimerize following recruitment into multiprotein complexs such as apotome or the death-inducing signaling complex. Once they activated, upstream Caspases undergo autocleavage, but dimerization rather than autocleavage is the primary activating event. Activated upstream caspases then cleave downstream procaspases. In contrast to upstream procaspases, downstream procaspases already exist as dimers. Instead, the activating event for downstream procaspases is cleavage, which separates the procaspase into three parts: prodomain, p20 subunit, and p10 subunit. The activated downstream Caspases is formed by the noncovalent reassembly of two p20 and two p10 subunits. The major role of downstream caspases is to cleave up to structural and regulatory, several hundred cellular proteins, to bring about cell death. The precise mechanisms by which this proteolytic cascade deconstructs the cell are incompletely understood

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